WHAT IS CLAIMED IS:

1. A method of inhibiting cell adhesion molecule cleavage in brain tissue of a host, said method comprising:

administering to said host an effective amount of a protease inhibitor.

2. The method according to Claim 1, wherein said protease inhibitor is a serine protease inhibitor.

3. The method according to Claim 1, wherein cell adhesion molecule cleavage in said brain tissue is associated with pathophysiology in said host.

4. The method according to Claim 1, wherein cell adhesion molecule cleavage in said brain tissue is associated with neuropathology in said host.

5. A method for treating a host for a pathological condition associated with cleavage of cell adhesion molecules in brain tissue, said method comprising:

administering to said host an effective amount of a protease inhibitor.

6. The method according to Claim 5, wherein said brain tissue is hippocampal tissue.

7. The method according to Claim 5, wherein said cell adhesion molecules are present on the surface of neurons.

8. The method according to Claim 5, wherein said cleavage is extracellular.

9. The method according to Claim 5, wherein said protease is a serine protease.

10. The method according to Claim 9, wherein said cell adhesion molecule has an extracellular domain comprising the residue sequence A-S-L-A and close relatives thereof.

11. The method according to Claim 10, wherein said host is a mammalian host.

12. The method according to Claim 5, wherein said host is suffering from a seizure, stroke, cerebral trauma or cerebral ischemia.

13. A method for treating a mammalian host for a pathological condition resulting at least in part from proteolysis of the extracellular domains of cell adhesion molecules, said method comprising:

administering to said host an effective amount of a serine protease inhibitor.

14. The method according to Claim 13, wherein said proteolysis results from excessive glutamate receptor activity.

15. The method according to Claim 13, wherein said glutamate receptor is an NMDA-type glutamate receptor.

16. The method according to Claim 14, wherein said proteolysis results in neuronal demise.

17. The method according to Claim 14, wherein said excessive glutamate receptor activity results from an event selected from the group consisting of stroke, head trauma and hypoxia.

18. The method according to Claim 17, wherein said proteolysis results in long-term potentiation.

19. The method according to Claim 17, wherein said proteolysis results in gains in excitatory responsiveness.

20. The method according to Claim 18, wherein said pathological condition is characterized by the presence of seizures.

21. The method according to Claim 20, wherein said pathological condition is epilepsy.

22. The method according to Claim 13, wherein said serine protease is a trypsin-like-serine protease.

23. The method according to Claim 22, wherein said inhibitor is a trypsin-like-serine protease inhibitor.

24. The method according to Claim 23, wherein said inhibitor is a tPA inhibitor.

25. The method according to Claim 24, wherein said inhibitor is AEBSF, and mimetics thereof.